his senses, he goes to bed without noticing the cut, probably rubs it with a dirty hand, and does not cover it up or wash it. Streptococci therefore gain entrance, and are not forthwith ejected. So they multiply: the leucocytes are called up, and attempt to destroy them, or, failing that, to form themselves into a barrier between the germs and the blood vessels of the part, in order to bar their entrance to the system. I want you to bear in mind that, in wound infection, they are always trying to do this: like the Communists in Paris, they erect barricade after barricade; driven from one, they fight at the next, and so on.

Behind these barricades more leucocytes are constantly being sent up as reinforcements, and at any stage the barrier wall may be effectual. Meanwhile the organisms are firing away at the obstruction with toxins, which will cause damage—it may be, death—if they get through into the general circulation. What happens to the patient depends upon the amount of un-neutralised toxin that obtains entrance to the blood stream, and that is determined, as I say, by the efficacy of the fighting powers of the patient's leucocytes.

Let us come back to our cut head. His leucocytes are probably not in good fighting trim, as they are partially paralysed by the alcohol, so the first few barricades are not of much use. The leucocytes are fighting hand to hand with the germs all over the skin near the wound: this becomes inflamed, as we say, and we have an attack of erysipelas, which is not by-the-bye, a specific disease, but just an inflammation of the skin due to streptococci. As each barricade is broken down, we see the result clinically in the spread of the erysipelatous blush on the skin. In some cases, this spreads over almost the whole body, but, as a rule, it becomes limited after a short time, and the patient recovers. The effects of the toxins already absorbed are shown in the fever and delirium from which the patient suffers; perhaps, also, there are attacks of shivering, with a high temperature which we know as rigors, and these are in any septic disease a bad sign.

Generally, patients recover from erysipelas. Whether they do so or not, depends, as I have said, on the amount of free toxin in the blood, and this again is determined mainly by the patient's powers of resistance. The reason why erysipelas is not usually fatal is that the organisms do not get deeper than the skin itself, so their field is somewhat limited, but when the resistance is lowered by such factors as alcoholism, kidney disease, or starvation—to take the commonest—the patient may succumb.

Let us now imagine that the germs have penetrated a little deeper, and we will take the case of a nurse who has pricked her finger and taken no notice of it. Here we shall see little or nothing wrong with the part itself at first, but it soon becomes swollen by something stretching the skin from underneath, and the finger may be painful, especially on pressure. The next thing is that she feels ill, perhaps shivers, and has a bad headache. Her temperature is taken, and is found to be 102 degs. or more. Obviously, there is toxin circulating, and the local swelling shows that the fight is going on just underneath the skin, in the cellular tissue, in fact.

Now, this inflammation of the cellular tissue—cellulitis, as it is called—is a more serious matter than the erysipelas of our alcoholic friend, simply because the battlefield is not in a hard organ like the skin, where limitation is easy, but in a loose region, where there is obviously much more room for the organisms and their toxins to run about—it is the difference, if you like, between fighting in a town and in a broad field; it is much harder to erect barricades in the open.

And so it happens in practice. In cellulitis, the spread is much more rapid. In a few hours only, the whole arm may be swollen, red and hard, and if we cut into the cellular tissue we find it soaked with a fluid, which may look like pus, or not, but, at all events, is seen, under the microscope, to consist of streptococci and leucocytes together, some alive and many dead. Consequently, the signs of toxaemia are more intense, rigors are more common, and death in two or three days not infrequently occurs. If we cut into the affected part after death we find the tissues just underneath the skin dead and gangrenous, like the ruined villages left in the track of a victorious army in an enemy's country. As a matter of fact, an attack of cellulitis is almost invariably fatal if not treated surgically, and, with the best intentions in the world, we often find our surgery has not been sufficiently extensive to check the disease.

There is yet a third variety of infection, in which the organisms are implanted deeper still, as when a septic ligature is left in the innermost parts of a wound, or, it may be, in some cavity like the abdomen. In such a case the leucocytes surround the offending article as before, and, while those nearest the organisms may succumb, the remainder survive and shut in the germs, so that an abscess is formed, which gradually works its way to the surface of the skin, or "points," as we say. Failing this, if the barrier is very effectual, the

previous page next page